MÉNIÈRE'S DISEASE

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BEFORE discussing sympathectomy I would like to say a few words about another form of conservative surgical treatment; that is to say conservative in the sense that hearing can often be preserved, though surgically it is a much bigger affair than those already described: I refer to intracranial section of the vestibular part only of the VIIIth nerve. This was first described by McKenzie of Toronto (1932). Then Dandy of Baltimore (1937), and more recently Olivecrona of Stockholm have reported large series of cases successfully treated in this way.

Being an intracranial operation and in particular being in the region of the cerebello-pontine angle, it is a major procedure that is not entirely without risk. However, it is claimed that in at least 75 per cent. of the cases the hearing is spared and my experience of patients so operated upon at the National Hospital for Nervous Diseases, Queen Square, by my colleagues, Mr. Harvey Jackson and Mr. Wylie McKissock, has been that in most the hearing was spared.

Because it is a more serious undertaking than operations on the labyrinth itself, intracranial section of the vestibular nerve is usually reserved for unilateral cases with good hearing on the affected side, and in bilateral cases where one side only is active and where the hearing is still useful.

Now I would like to say a few words about the condition which we are trying to treat; and though we do not know the cause we do have some knowledge of what effect it has upon the labyrinth, and this encourages us to speculate upon what happens inside the endolymphatic labyrinth during the active phase of the disease. Until 1938 haemorrhage from a ruptured vessel or anæmia in consequence of vascular thrombosis were often held responsible for the disorder; though Knapp and Cheatle more than 50 years ago suggested that an increase of endolymphatic pressure analogous to glaucoma might be the cause, and more recently Mygind and Dederding (1932) regarded it as a disorder of water metabolism.

Then in 1938 Hallpike and Cairns were able to show that distension of the cochleo-saccular part of the endolymphatic system was the visible result of Ménière's disease. This they attributed to rising pressure within the endolymphatic system as the result of inability to dispose of endolymph as quickly as it is formed. They accepted Guild's view on the origin

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circulation and disposal of the fluid endolymph suggesting that the fault lay in the region of the saccus endolymphaticus.

Since then the fluctuations in endolymphatic pressure with variations in cochlear and vestibular symptoms which go to make up Ménière's disease have been explained on a variety of grounds, including arteriolar spasm, capillary stasis and exudation, venous congestion, allergic or infective œdema and herpetic vesiculation; to mention some of the views put forward, sometimes to justify a particular form of treatment.

Now, as we still are not sure of the way in which the delicate endolymphatic system works, we cannot for certain confirm or deny any of these mechanisms, and in the presence of an inefficient pressure regulating mechanism these and other factors may play some part in precipitating attacks.

What we do know, however, is that these fluctuations in endolymphatic pressure are by far the commonest expression of a labyrinthine disorder; for Ménière's disease accounts for many more cases of aural vertigo than all the other causes put together as the following table shows.

In this table are included all the cases of vertigo which I have seen in the past 20 years and of which I have some record.

TABLE I.

CAUSES OF VERTIGO IN A SERIES OF 2,174 CASES.

CENTRAL		201
PERIPHERAL		1,833
Ménière's Disease	1,278	
Vestibular Neuronitis	183	
Peripheral Positional	144	
Trauma	146	
Infective	82	
NOT CLASSIFIED		140

You will note that of peripheral causes 70 per cent. are due to Ménière's disease. In other words this one cause accounts for more than twice all the rest put together.

Now, surely there must be some significance in this overwhelming preponderance of one disorder.

If, as is generally believed, the attacks are due to a defect in the pressure regulating mechanism of the endolymphatic system, then one is irresistibly drawn to the conclusion that Ménière's disease is the consequence of an inherent weakness in that system.

Many organs or systems in the body have their weak spot which appears as the result of wear and tear, and the pressure regulating mechanism seems to be the most vulnerable part of the endolymphatic system.

It will, at this stage, be convenient to consider what happens to the endolymph.

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According to Guild (1927), the endolymph is a circulating fluid quite separate from the perilymphatic and cerebrospinal fluid systems. He believes that the endolymph is secreted by the stria vascularis of the cochlea, that it circulates through the membranous labyrinth and escapes possibly by being pumped out through the wall of the saccus endolymphaticus into the subdural space.

Now, if for some reason or other there is delay in the final disposal of the endolymph through the wall of the saccus, or, indeed, if there is a hold-up anywhere in the system, then there will be a building up of pressure within that part of the endolymphatic system proximal to the obstruction. This will affect the sensory end-organs. If the pressure rises enough to stop the flow of blood through the capillaries, the sensory end-organs will suddenly cease to respond. It is unlikely that these delicate end-organs can survive if they are deprived of their blood supply for very long. For this reason it is suggested that the rising pressure within the endolymphatic system will tend to slow down the secretion of more endolymph until perhaps it stops altogether. Thus the pressure will subside as the surplus endolymph slowly drains away. The pressure may return to normal quite quickly or it may remain somewhat raised, with an occasional sudden increase causing an attack, over a period of weeks or months before it finally subsides.

We do not know whether this state of affairs is likely to be brought about by mechanical means in the pumping out mechanism, whether one of the fine communicating tubes becomes blocked in some way or other, or whether there is some variation in the physico-chemical constitution of the endolymph; but clearly whatever the cause, it can operate in one labyrinth only, as the disease is usually unilateral.

It is unlikely that the endolymphatic pressure is ever great enough to cause dilatation of the whole vestibular labyrinth; for before the relatively firm walls of the membranous canals can dilate the thinner membranes in the cochlea and saccule will rupture with subsequent loss of both cochlear and vestibular function.

We have met with cases in which after one severe attack there has been total loss of function; and this has often been attributed to a vascular disaster. True, such a disaster could happen, but the general behaviour of the disease suggests that rupture of the endolymphatic system may also happen. The foregoing suggestion of the events taking place inside the labyrinth during the active phase of Ménière's disease does help to explain the symptoms and signs of the disorder.

And now, at last, we come to the subject of today's discussion, namely Conservative Surgery in the Treatment of Ménière's Disease.

At once, some procedure that will encourage a freer flow of endolymph from the saccus endolymphaticus springs to the mind, and indeed I. Portmann, as long ago as 1927, incised the saccus for the relief of Ménière's

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disease. Logical though this operation seems, the results in most hands have not been sufficiently encouraging for it to be generally practised nowadays.

I did five of these operations some years ago and, despite using magnification, I found that in some it was difficult to be sure that one had really incised the saccus. In view of what we now know, any substantial draining off of endolymph might be expected to result in loss of vestibular and cochlear function. Three of the five patients on whom I operated had subsequently to have the labyrinth destroyed; the other two could not be traced. On thinking it over one cannot help feeling that the scar and fibrous tissue that must inevitably follow surgery, may prove to be an additional hindrance to the normal escape of endolymph.

2. Limited operations on the vestibular labyrinth often succeed in destroying both cochlear and vestibular function. If some function is retained, it may well happen that vestibular as well as cochlear function is spared.

3. Now we come to the effect of stellate ganglionectomy on the progress of Ménière's disease. In 1949 and 1951 we had interesting and enthusiastic papers about this operation from the late Garnett Passe and Seymour, and after their last paper Roland Lewis and I felt that we should give it a trial. You have already heard from him and Harrison about the results. I should add that Lewis did all the operations in the King's series and I referred some of the subjects to him. Again, it is only fair to admit that many of the cases I referred to him were really difficult problems, often with bilateral disease which had defied all attempts at conservative treatment. I have recently reviewed all these cases quite independently of Lewis and our combined findings have been given under the heading of "King's cases".

Having seen the sorry plight of some of the patients before operation, in contrast with their present contented and settled state, I am quite sure that the operation must have done something to bring about this change. Now, one of the interesting things is that the change rarely took place immediately after the operation. In fact, several patients had quite severe attacks within a few days of the operation, but subsequently did very well.

Thus it seems that whatever effect stellate ganglionectomy has upon the internal ear of sufferers from Ménière's disease, it often does not act at once and it sometimes modifies rather than abolishes the attacks, while in a minority of the cases (about one quarter) it does not have any effect at all.

We have found that injection of the stellate ganglion with xylocaine does not often have any influence on the course of the disease, nor does it seem to give any definite indications as to which cases may be helped by stellate ganglionectomy.

The difficulty in assessing the value of any conservative treatment

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is the natural tendency of Ménière's disease to periods of freedom from attacks, periods which may extend over years. The physician who admitted that he owed his success to a knack of anticipating Nature's tendency to cure might well have been thinking of Ménière's disease.

Only last month a patient said to me that six years previously she had been troubled with a succession of bouts of giddiness, which finally stopped after two sessions with a faith-healer, only to return five years later with undiminished severity. When asked why she had not returned to the faith-healer she replied, "Because I have always felt that it stopped on its own."

In view of all this I thought it would be interesting to see what happened to a random group of patients seen during the first half of 1952. The only selection has been to exclude any about whom we have insufficient knowledge of their progress.

TABLE II.

FORTY CASES OF MÉNIÈRE'S DISEASE FIRST SEEN IN 1952.

No attacks for 2 years or more	15
Attacks within past 2 years	13
Operation	12

Thus 37·5 per cent. of this admittedly small group have remained free of symptoms for two years or more on simple, non-surgical conservative treatment, or in one or two cases I believe no treatment at all.

Those operated upon all had stubborn attacks which were entirely relieved by labyrinthectomy in 10 cases and vestibular nerve section in 2.

Now in all fairness I must say that this group does not include as many bilateral and otherwise problem cases as those in Lewis's series. Most of his were difficult cases and I have been favourably impressed by the improvement brought about as the result of stellate ganglionectomy.

We feel that it deserves further trial, and in particular we would like to see how it will effect the progress of early cases, as well as in any other cases in which preservation of cochlear function is desirable.

You may well think that it is always desirable to preserve cochlear function, but I must draw your attention to the type of case in which only one ear is affected, and that only a disordered and useless remnant of hearing is present. In such a case simple destruction of labyrinthine function is indicated.

Again, in a unilateral case when it is essential for the well-being of the patient that the attacks should cease, I am thinking of patients who are demoralized by frequent attacks or who are in danger of losing employment if the attacks continue, then some more certain form of operation than stellate ganglionectomy will be needed, either labyrinthectomy or vestibular nerve section.

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I have not said anything about the application of ultrasonic frequencies to the vestibular labyrinth as advocated by my friend Professor Arslan as I have not, as yet had any personal experience of this interesting new method, but we shall all look forward to hearing more about this in the future.

I think that future work should include further investigation into the possibility of using streptomycin in such a way that the vestibular neurones on the diseased side only are affected by the drug.

And now I would like to leave you with the thought that Ménière's disease may be the Achilles heel of the endolymphatic system, a system about which we still have a great deal to learn.

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